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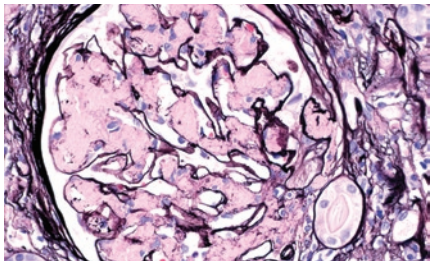
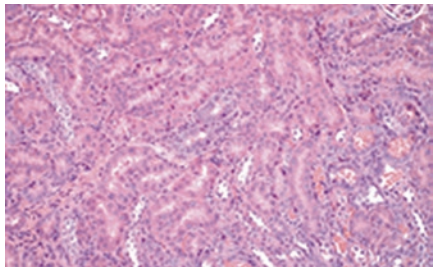
LECT2, a new amyloid component



Some patients with amyloidosis have deposits that do not react to antibodies that recognize the traditional causes of amyloidosis, such as immunoglobulin light chains-λ and -κ and serum amyloid A. These deposits react with Congo red; hence, it is obvious that there are additional proteins that have the classic β-sheets that can make amyloid deposits. As they report in this issue, Larsen *et al.* isolated amyloid fibrils from such patients, subjected them to chemical analysis, and found leukocyte chemotactic factor 2 (LECT2) to be the protein responsible for these deposits. From a large referral base with 285 renal amyloid samples, 31 could not be classified by the classic causes. Tandem mass spectrometry of these 31 samples showed that seven of them were LECT2, which was confirmed by immunohistochemistry. These deposits also stained for Congo red and, in most cases, had distinctive morphological features with diffuse involvement of the interstitium, arteries, and glomeruli. **See page 816.**

Regulatory T cells are protective in ischemic preconditioning

One of the most popular models of acute tubular necrosis is produced by induction of ischemia. Numerous studies have found that when renal blood flow is re-established, this reperfusion produces a large number of changes that, in fact, may be causal to the acute kidney injury. Remarkably, a short period of ischemia seems to protect the kidney from injury induced by a subsequent ischemia. As they report in this issue, Kinsey *et al.* used this so-called ischemic preconditioning, which is partially mediated by T regulatory (Treg) lymphocytes that suppress immune responses. The authors found that this maneuver inhibited the accumulation of neutrophils and macrophages, tubular necrosis, and loss of kidney function caused by a subsequent ischemia/reperfusion injury. Depletion of the Treg cells with an antibody reversed the beneficial effect of preconditioning on kidney neutrophil accumulation and partially inhibited the functional and histological



protection of preconditioning. Further, adoptive transfer of Treg cells in naive mice, before ischemia/reperfusion, mimicked the protective and anti-inflammatory effects of ischemic preconditioning on the kidney. These studies show that Treg cells are critical in protecting the kidney in this model of ischemic preconditioning. **See page 771.**

Tubulointerstitial disease in lupus nephritis

The system for classifying patients with lupus nephritis provided by the International Society of Nephrology and the Renal Pathology Society in 2003 rapidly became the gold standard in classification of the disease, and the basis for many studies and therapeutic decisions. However, the classification system was based entirely on glomerular lesions, even though it is well known that interstitial disease occurs in lupus nephritis. Yu *et al.* analyzed the tubulointerstitial lesions in patients with lupus nephritis. They found that among 313 patients, the 170 with class IV had severe interstitial inflammatory-cell infiltration, tubular atrophy, and interstitial fibrosis. Tubulointerstitial disease was present in almost all types of lupus glomerular disease, to varying degrees. These important studies show that the extent of tubulointerstitial lesions needs to be taken into account when the renal outcome in patients with lupus nephritis is estimated. **See page 820.**